10/049,320

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(FILE 'HOME' ENTERED AT 18:05:27 ON 23 SEP 2004)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 18:06:23 ON 23 SEP 2004

L1 965 S CD39
L2 77748 S (ISCHEMIC(W)DISORDER OR PERIPHERAL(W)VASCULAR OR PULMONARY(W)
L3 539394 S MYOCARDIAL(W)INFARCTION OR UNSTABLE(W)ANGINA OR ISCHEMIC(3A)(
L4 608284 S L2 OR L3
L5 20 S L1(9A)L4
L6 12 DUP REM L5 (8 DUPLICATES REMOVED)
L7 44 S L1 AND L4
L8 27 DUP REM L7 (17 DUPLICATES REMOVED)

=> d au ti so pi ab 1-12 l6

L6 ANSWER 1 OF 12 MEDLINE on STN DUPLICATE 1
AU Marcus A J; Broekman M J; Drosopoulos J H F; Islam N; Pinsky D J; Sesti C; Levi R
TI Heterologous cell-cell interactions: thromboregulation, cerebroprotection and cardioprotection by CD39 (NTPDase-1).
SO Journal of thrombosis and haemostasis : JTH, (2003 Dec) 1 (12) 2497-509. Ref: 59
Journal code: 101170508. ISSN: 1538-7933.
AB Blood platelets maintain vascular integrity and promote primary and secondary hemostasis following interruption of vessel continuity. Biochemical or physical damage to the coronary, carotid or peripheral arteries is followed by excessive platelet activation and recruitment culminating in vascular occlusion and tissue ischemia. Currently inadequate therapeutic approaches to stroke and coronary artery disease are a public health issue. Following our demonstration of neutrophil leukotriene production from arachidonate released from activated aspirin-treated platelets, we studied interactions between platelets and other blood cells, leading to concepts of transcellular metabolism and thromboregulation. Thrombosis has a proinflammatory component whereby biologically active substances are synthesized by interactions between different cell types that could not individually synthesize the product(s). Endothelial cells control platelet reactivity via three biochemical systems-autacoids leading to production of prostacyclin and nitric oxide, and endothelial ecto-ADPase/CD39/NTPDase-1. The autacoids are fluid-phase reactants, not produced by tissues in the basal state. They are only synthesized intracellularly and released upon interactions of cells with an agonist. When released, autacoids exert fleeting actions in the immediate milieu, and are rapidly inactivated. CD39 is an integral component of the endothelial cell surface and is substrate-activated. It maintains vascular fluidity in the complete absence of prostacyclin and nitric oxide, indicating that they are ancillary components of hemostasis. Therapeutic implications for the autacoids have not been compelling because of their transient, local and fleeting action, and limited potency. Conversely, CD39, acting solely on the platelet releasate, is efficacious in three different animal models. It metabolically neutralizes a prothrombotic platelet releasate via deletion of ADP--the major recruiting agent responsible for formation of an occlusive thrombus. In addition, solCD39 reduced ATP- and ischemia-induced norepinephrine release in the heart. This reduction can prevent fatal arrhythmia. Moreover, solCD39 ameliorated the sequelae of **stroke** in **CD39** null mice. CD39 represents the next generation of cardioprotective and cerebroprotective molecules.

L6 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 2
AU Marcus, Aaron J.; Broekman, M. Johan; Drosopoulos, Joan H. F.; Islam, Naziba; Pinsky, David J.; Sesti, Casilde; Levi, Roberto
TI Metabolic control of excessive extracellular nucleotide accumulation by

CD39/ecto-nucleotidase-1: Implications for ischemic vascular diseases
SO Journal of Pharmacology and Experimental Therapeutics (2003), 305(1), 9-16
CODEN: JPETAB; ISSN: 0022-3565
AB A review. Platelets are responsible for maintaining vascular integrity. In thrombocytopenic states, vascular permeability and fragility increase, presumably due to the absence of this platelet function. Chemical or phys. injury to a blood vessel induces platelet activation and platelet recruitment. This is beneficial for the arrest of bleeding (hemostasis), but when an atherosclerotic plaque is ulcerated or fissured, it becomes an agonist for vascular occlusion (thrombosis). Expts. in the late 1980s cumulatively indicated that endothelial cell CD39-an ecto-ADPase-reduced platelet reactivity to most agonists, even in the absence of prostacyclin or nitric oxide. As discussed herein, CD39 rapidly and preferentially metabolizes ATP and ADP released from activated platelets to AMP, thereby drastically reducing or even abolishing platelet aggregation and recruitment. Since ADP is the final common agonist for platelet recruitment and thrombus formation, this finding highlights the significance of CD39. A recombinant, soluble form of human CD39, solCD39, has enzymic and biol. properties identical to the full-length form of the mol. and strongly inhibits human platelet aggregation induced by ADP, collagen, arachidonate, or TRAP (thrombin receptor agonist peptide). In sympathetic nerve endings isolated from guinea pig hearts, where neuronal ATP enhances norepinephrine exocytosis, solCD39 markedly attenuated norepinephrine release. This suggests that NTPDase (nucleoside triphosphate diphosphohydrolase) could exert a cardioprotective action by reducing ATP-mediated norepinephrine release, thereby offering a novel therapeutic approach to myocardial ischemia and its consequences. In a murine model of stroke, driven by excessive platelet recruitment, solCD39 reduced the sequelae of stroke, without an increase in intracerebral hemorrhage. CD39 null mice, generated by deletion of apyrase-conserved regions 2 to 4, exhibited a decrease in postischemic perfusion and an increase in cerebral infarct volume when compared with controls. "Reconstitution" of CD39 null mice with solCD39 reversed these changes. We hypothesize that solCD39 has potential as a novel therapeutic agent for thrombotic diatheses.

L6 ANSWER 3 OF 12 MEDLINE on STN DUPLICATE 3
AU Pinsky David J; Broekman M Johan; Peschon Jacques J; Stocking Kim L; Fujita Tomoyuki; Ramasamy Ravichandran; Connolly E Sander Jr; Huang Judy; Kiss Szilard; Zhang Yuan; Choudhri Tanvir F; McTaggart Ryan A; Liao Hui; Drosopoulos Joan H F; Price Virginia L; Marcus Aaron J; Maliszewski Charles R
TI Elucidation of the thromboregulatory role of CD39/ectoapyrase in the ischemic brain.
SO Journal of clinical investigation, (2002 Apr) 109 (8) 1031-40.
Journal code: 7802877. ISSN: 0021-9738.
AB Endothelial CD39 metabolizes ADP released from activated platelets. Recombinant soluble human CD39 (solCD39) potently inhibited ex vivo platelet aggregation in response to ADP and reduced cerebral infarct volumes in mice following transient middle cerebral artery occlusion, even when given 3 hours after stroke. Postischemic platelet and fibrin deposition were decreased and perfusion increased without increasing intracerebral hemorrhage. In contrast, aspirin did not increase postischemic blood flow or reduce infarction volume, but did increase intracerebral hemorrhage. Mice lacking the enzymatically active extracellular portion of the CD39 molecule were generated by replacement of exons 4-6 (apyrase-conserved regions 2-4) with a PGKneo cassette. Although CD39 mRNA 3' of the neomycin cassette insertion site was detected, brains from these mice lacked both apyrase activity and CD39 immunoreactivity. Although their baseline phenotype, hematological profiles, and bleeding times were normal, cd39(-/-) mice exhibited increased cerebral infarct volumes and reduced postischemic perfusion. solCD39 reconstituted these mice, restoring postischemic cerebral perfusion and rescuing them from cerebral injury. These data demonstrate

that **CD39** exerts a protective thromboregulatory function in **stroke**.

L6 ANSWER 4 OF 12 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU El-Omar, Magdi M. [Reprint Author]; Islam, Naziba; Broekman, M. Johan; Drosopoulos, Joan H. F.; Roa, Donald C.; Lorin, Jeffrey; Sedlis, Steven P.; Marcus, Aaron J.

TI ATP/ADP Ectonucleotidase Activity Is Increased in Patients with Coronary Artery Disease.

SO Blood, (November 16 2002) Vol. 100, No. 11, pp. Abstract No. 1932. print. Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.

AB CD39 (E-NTPDase-1) is the predominant ectonucleotidase at the luminal surface of blood vessels as well as on lymphocytes. Recent evidence from our group indicates that CD39 plays a key role in thromboregulation by hydrolyzing ADP released from activated platelets, while also metabolizing ATP at a similar rate. In a murine model of **stroke**, endogenous and/or exogenously administered **CD39** limited the sequelae of **stroke**. The higher the ratio of ATPase/ADPase activity of an ectonucleotidase, the lower its platelet anti-aggregatory effect. In the present study we examined ectonucleotidase ATPase and ADPase activities in patients with coronary artery disease (CAD). Males with angiographically-documented CAD (gtoreq1 major vessel with gtoreq50% stenosis) were compared to a group of age-matched healthy males without apparent CAD (controls). Lymphocytes were isolated from heparinized whole blood using Histopaque gradient centrifugation, and washed prior to incubation with 50muM 14C ADP or 14C ATP (5 min, 37degreeC). Thin layer chromatography (TLC) was used to separate nucleotides, nucleosides and bases, and radioactivity was quantified by radio-TLC scanning. Data were expressed as pmoles ATP or ADP metabolized per min per 5x104 lymphocytes, and ATPase/ADPase activity ratios were calculated. Results: ATPase activity was higher in CAD compared to controls, despite similar ADPase activity. The ratio of ATPase/ADPase activity was significantly higher (apprx20%, $p<0.005$) in CAD compared to controls (Table). Conclusion: Patients with CAD have an increased ATP/ADP ectonucleotidase activity ratio, possibly due to an alteration in CD39 nucleotide specificity and/or co-expression of another ectonucleotidase with greater ATP specificity. The altered ATPase/ADPase activity ratio may lower endogenous defenses against platelet-driven thrombotic events in these patients. The results suggest that NTPDase-1 or recombinant derivatives therefrom may represent a novel therapeutic modality to occlusive vascular diseases.

L6 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN

IN Pinsky, David J.

TI Method using **CD39**/ecto-ADPase for treating thrombotic and **ischemic disorders**

SO PCT Int. Appl., 119 pp.
CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001011949	A1	20010222	WO 2000-US22060	20000811
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002138858	A1	20020926	US 1999-374586	19990813

AB The present invention provides a method of treating or preventing thrombotic or **ischemic disorders** in a subject which comprises administering an **CD39**/ecto-ADPase to the subject, wherein the CD39/ecto-ADPase inhibits ADP-mediated platelet aggregation by increasing ADP catabolism, and a method for determining whether a compound inhibits platelet aggregation by increasing ADP catabolism so as to treat or prevent thrombotic or ischemic disorders in a subject, comprising: (a) inducing thrombotic or ischemic disorders in an animal, which animal is an animal model for thrombotic or ischemic disorders; (b) measuring the stroke outcome in said animal; (c) measuring platelet deposition and/or fibrin deposition in ischemic tissue; and (d) comparing the stroke outcome in step (b) and the platelet deposition and/or fibrin deposition with that of the animal model in the absence of the compound so as to identify a compound capable of treating or preventing thrombotic or ischemic disorders in a subject. Also disclosed are human CD39/ecto-ADPase sequences.

L6 ANSWER 6 OF 12 MEDLINE on STN

AU Marcus A J; Broekman M J; Drosopoulos J H; Pinsky D J; Islam N; Maliszewsk C R

TI Inhibition of platelet recruitment by endothelial cell CD39/ecto-ADPase: significance for occlusive vascular diseases.

SO Italian heart journal : official journal of the Italian Federation of Cardiology, (2001 Nov) 2 (11) 824-30.
Journal code: 100909716. ISSN: 1129-471X.

AB During their 7-9 day lifespan in the circulation platelets are mainly responsible for maintaining the integrity of the vasculature. In thrombocytopenic states, there is an increase in vascular permeability and fragility, presumably due to absence of this platelet function. In sharp contrast, biochemical or physical injury in the coronary, carotid or peripheral arteries induces platelet activation and platelet recruitment, which can culminate in thrombotic vascular occlusion. Since there is one death every 33 s from vascular occlusion in the United States, this situation constitutes a major public health issue. In the course of studying interactions between cells of the vascular wall and those in the circulation, we observed that platelets in close proximity to endothelial cells do not respond to agonists in vitro. Experiments initiated in the late 1980's cumulatively indicated that endothelial cell CD39--an ecto-ADPase--was mainly responsible for this phenomenon. CD39 rapidly and preferentially metabolizes ADP released from activated platelets. ADP is the final common pathway for platelet recruitment and thrombus formation, and platelet aggregation and recruitment are abolished by CD39. Our current hypothesis is that CD39 will be a novel antithrombotic agent for treating high risk patients who have activated platelets in their circulation--the identifying characteristic of coronary artery occlusion and thrombotic stroke. A recombinant, soluble form of human CD39 has been generated. This is solCD39, a glycosylated protein of 66 kDa whose enzymatic and biological properties are identical to the full-length form of the enzyme. In our in vitro experiments, solCD39 blocks ADP-induced human platelet aggregation, and inhibits collagen- and thrombin receptor agonist peptide-induced platelet reactivity. We studied solCD39 in vitro in a murine model of stroke, which was shown to be driven by excessive platelet recruitment. In studies with CD39 wild-type (CD39+/+) mice solCD39 completely abolished ADP-induced platelet aggregation, and strongly inhibited collagen- and arachidonate-induced platelet reactivity ex vivo. When solCD39 was administered prior to transient intraluminal middle cerebral artery occlusion, it reduced ipsilateral fibrin deposition, decreased (111)In-platelet deposition, and increased post-ischemic blood flow 2-fold at 24 hours. These results were superior to those we obtained with aspirin pre-treatment. CD39 null (CD39-/-) mice, which we generated by deletion of exons 4-6 (apyrase conserved regions 2-4), have a normal phenotype, normal hematologic profiles and bleeding times, but exhibit a decrease in post-ischemic perfusion and an increase in cerebral infarct volume when compared to genotypic **CD39**+/+ controls in our **stroke** model. "Reconstitution"

of **CD39** null mice with solCD39 reversed these pathologic changes. Thus, the CD39-/- mice were actually rescued from cerebral injury by solCD39, thereby fulfilling Koch's postulates. These experiments have led us to hypothesize that solCD39 has potential as a novel therapeutic agent for thrombotic stroke. In this review, we summarize our recent research results with CD39 and solCD39, and discuss our viewpoints on its present and future possibilities as a novel treatment for thrombosis.

L6 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
AU Weksler, Babette B.; Pena-Alvarez, Jose
TI New antiplatelet strategies in stroke prevention and treatment
SO Drug Therapy for Stroke Prevention (2001), 94-110. Editor(s): Bogousslavsky, Julien. Publisher: Taylor & Francis Ltd., London, UK. CODEN: 69EDI6; ISBN: 0-7484-0934-3
AB A review. A continued search for more effective antiplatelet therapy appears important for improving the prevention and treatment of occlusive stroke, since current therapies are of limited benefit compared with efficacy of antiplatelet approaches to acute cardiac events. Measures that decrease the classic risk factors for cerebral ischemia, which often involve increased platelet reactivity, form the backbone of preventive strategies. While aspirin remains the best agent for patients who have had previous TIA and stroke, its benefits are modest (<25 % risk reduction in secondary prevention and about 12 % risk reduction in acute stroke) and combining aspirin with other known antithrombotics may well elevate risk of intracranial hemorrhage. Truly new approaches remain in the preclin. stage overall at the present time. Usage of platelet GPIIbIIIa antagonists is effective in reducing stroke damage in many animal models but has not yet been examined in clin. trials of stroke, in part because of concern about bleeding risk. Measures to block iNOS and to utilize NO donors to enhance endothelial NO production have also been shown to prevent brain damage and improve cerebral blood flow after exptl. cerebral ischemia; techniques to target N0-interventional strategies to areas of vascular damage are being developed. The soluble, recombinant form of the endothelial ectoADPase, CD39, which has antiplatelet effects in vitro, has been used in a mouse model of acute stroke where its infusion shows promising efficacy in reducing infarct size and restoring cerebral blood flow without incurring hemorrhage. Further development and clin. application of such novel measures are awaited with great interest.

L6 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
IN Maliszewski, Charles R.; Gayle, Richard B., III; Price, Virginia L.; Gimpel, Steven D.
TI CD39 polypeptides as inhibitors of platelet activation and recruitment
SO PCT Int. Appl., 122 pp.
CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000023459	A1	20000427	WO 1999-US22955	19991013
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2345382	AA	20000427	CA 1999-2345382	19991013
AU 9964115	A1	20000508	AU 1999-64115	19991013
AU 772460	B2	20040429		
EP 1123306	A1	20010816	EP 1999-951733	19991013
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

	JP 2002527096	T2	20020827	JP 2000-577185	19991013
	NZ 511488	A	20031031	NZ 1999-511488	19991013
	US 2002002277	A1	20020103	US 2001-835147	20010413

AB The present invention provides soluble CD39 polypeptides and compns., and methods for inhibiting platelet activation and recruitment in a mammal comprising administering a soluble CD39 polypeptide.

L6 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
IN Maliszewski, Charles R.; Gayle, Richard B., III; Marcus, Aaron J.
TI Methods of inhibiting platelet activation and recruitment
SO PCT Int. Appl., 118 pp.
CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000023094	A2	20000427	WO 1999-US23641	19991013
WO 2000023094	A3	20000727		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

	AU 9964256	A1	20000508	AU 1999-64256	19991013

AB The present invention provides soluble CD39 polypeptides and compns., and methods for inhibiting platelet activation and recruitment in a mammal comprising administering a soluble CD39 polypeptide.

L6 ANSWER 10 OF 12 MEDLINE on STN DUPLICATE 4
AU Weksler B B
TI Antiplatelet agents in stroke prevention. combination therapy: present and future.
SO Cerebrovascular diseases (Basel, Switzerland), (2000) 10 Suppl 5 41-8.
Ref: 43
Journal code: 9100851. ISSN: 1015-9770.

AB Platelets contribute to arterial thrombosis by multiple mechanisms that promote blood clotting, favor vasoconstriction, activate the procoagulant capacity of endothelium, and stimulate inflammation. These activities are augmented by turbulent blood flow. Classic antiplatelet therapy with aspirin to prevent occlusive stroke offers significant clinical benefit (20-25% risk reduction), yet is less effective than in prevention of coronary artery occlusion (up to 50% risk reduction of myocardial infarction in unstable angina). Since aspirin's antiplatelet effects are limited to blocking a single metabolic pathway - namely inhibition of thromboxane A(2) formation -, and aspirin fails to alter platelet adhesion, other antiplatelet agents that target ADP receptors, platelet surface glycoproteins (such as the GPIIb/IIIa complex), or platelet-dependent thrombin generation offer additional clinical benefits by blocking additional separate pathways or the final common pathway of platelet activation. Combinations of antiplatelet agents, such as aspirin/dipyridamole, aspirin/clopidogrel, or aspirin/GPIIb/IIIa inhibitors, have recently been tested for improved efficacy in clinical trials. Soluble recombinant **CD39**, an ecto-ADPase, protects against **stroke** in animal models by metabolizing released ADP/ATP to antiplatelet derivatives. In general, combinations of antiplatelet agents promise greater efficacy than single drugs in preventing stroke, since interactions among different antiplatelet mechanisms can be synergistic. However, such combinations may also increase the risk of bleeding, so that precise understanding of risk/benefit ratios that address the possibility of intracranial as well as gastrointestinal bleeding will require careful monitoring in large clinical trials of patients at risk of stroke, with particular attention to the elderly.

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L6 ANSWER 11 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU McTaggart R A (Reprint); Broekman J; Peschon J; Stocking K; Choudhri T F; Kim L J; Connolly E S; Drosopoulos J H F; Maliszewski C R; Marcus A J; Pinsky D J
TI Cerebroprotective role of **CD39** (endothelial EctoADPase) in murine **stroke**
SO CIRCULATION, (2 NOV 1999) Vol. 100, No. 18, Supp. [S], pp. 1720-1720. Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621.
ISSN: 0009-7322.

L6 ANSWER 12 OF 12 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU McTaggart, Ryan A. [Reprint author]; Broekman, M. Johan; Peschon, Jacques; Stocking, Kim; Choudhri, Tanvir F.; Kim, Louis J.; Connolly, E. Sander, Jr.; Drosopoulos, Joan H. F.; Maliszewski, Charles R.; Marcus, Aaron J.; Pinsky, David J.
TI Cerebroprotective role of **CD39** (endothelial ectoADPase) in murine **stroke**.
SO Circulation, (Nov. 2, 1999) Vol. 100, No. 18 SUPPL., pp. I.328. print.
Meeting Info.: 72nd Scientific Sessions of the American Heart Association. Atlanta, Georgia, USA. November 7-10, 1999.
CODEN: CIRCAZ. ISSN: 0009-7322.

=> d au ti so 11-27 l8

L8 ANSWER 11 OF 27 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Qawi, Imrana [Reprint author]; Robson, Simon C. [Reprint author]
TI New developments in anti-platelet therapies: Potential use of **CD39** /vascular ATP diphosphohydrolase in thrombotic disorders.
SO Current Drug Targets, (June, 2001) Vol. 2, No. 2, pp. 213-214. print.
ISSN: 1389-4501.

L8 ANSWER 12 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN
IN Pinsky, David J.
TI Method using **CD39**/ecto-ADPase for treating thrombotic and **ischemic disorders**
SO PCT Int. Appl., 119 pp.
CODEN: PIXXD2

L8 ANSWER 13 OF 27 MEDLINE on STN
AU Marcus A J; Broekman M J; Drosopoulos J H; Pinsky D J; Islam N; Maliszewsk C R
TI Inhibition of platelet recruitment by endothelial cell **CD39** /ecto-ADPase: significance for occlusive vascular diseases.
SO Italian heart journal : official journal of the Italian Federation of Cardiology, (2001 Nov) 2 (11) 824-30.
Journal code: 100909716. ISSN: 1129-471X.

L8 ANSWER 14 OF 27 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Kiss S (Reprint); Marcus A J; Broekman M J; Nair M N; D'Ambrosio A L; Liao H; Maliszewski C R; Connolly E S; Pinsky D J
TI Soluble **CD39** but not aspirin decreases platelet deposition and improves outcome in reperfused murine **stroke**
SO STROKE, (JAN 2001) Vol. 32, No. 1, pp. 359-359. MA P109.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0039-2499.

L8 ANSWER 15 OF 27 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Levi, Roberto [Reprint author]; Koyama, Motohiro [Reprint author]; Sesti, Casilde [Reprint author]; Broekman, Marinus J.; Drosopoulos, Joan H. F.; Islam, Naziba; Marcus, Aaron J.
TI Protective effect of ecto-nucleotidases: Modulation of ATP-induced norepinephrine release during myocardial ischemia.
SO Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 248a. print. Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1. Orlando, Florida, USA. December 07-11, 2001. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.

L8 ANSWER 16 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN
AU Pinsky, David J.
TI New endothelium-based approaches to **stroke** therapy
SO NATO Science Series, Series I: Life and Behavioural Sciences (2001), 330(Vascular Endothelium), 100-107
CODEN: NSSSC9; ISSN: 1566-7693

L8 ANSWER 17 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN
AU Weksler, Babette B.; Pena-Alvarez, Jose
TI New antiplatelet strategies in **stroke** prevention and treatment
SO Drug Therapy for Stroke Prevention (2001), 94-110. Editor(s): Bogousslavsky, Julien. Publisher: Taylor & Francis Ltd., London, UK.
CODEN: 69EDI6; ISBN: 0-7484-0934-3

L8 ANSWER 18 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN
IN Maliszewski, Charles R.; Gayle, Richard B., III; Price, Virginia L.; Gimpel, Steven D.
TI **CD39** polypeptides as inhibitors of platelet activation and recruitment
SO PCT Int. Appl., 122 pp.
CODEN: PIXXD2

L8 ANSWER 19 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN
IN Maliszewski, Charles R.; Gayle, Richard B., III; Marcus, Aaron J.
TI Methods of inhibiting platelet activation and recruitment
SO PCT Int. Appl., 118 pp.
CODEN: PIXXD2

L8 ANSWER 20 OF 27 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Yeung G; Mulero J J; McGowan D W; Bajwa S S; Ford J E (Reprint)
TI CD39L2, a gene encoding a human nucleoside diphosphatase, predominantly expressed in the heart
SO BIOCHEMISTRY, (24 OCT 2000) Vol. 39, No. 42, pp. 12916-12923.
Publisher: AMER CHEMICAL SOC, 1155 16TH ST, NW, WASHINGTON, DC 20036.
ISSN: 0006-2960.

L8 ANSWER 21 OF 27 MEDLINE on STN DUPLICATE 6
AU Qawi I; Robson S C
TI New developments in anti-platelet therapies: potential use of **CD39** /vascular ATP diphosphohydrolase in thrombotic disorders.
SO Current drug targets, (2000 Nov) 1 (3) 285-96. Ref: 59
Journal code: 100960531. ISSN: 1389-4501.

L8 ANSWER 22 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN
AU Robson, Simon C.; Sevigny, Jean; Imai, Masato; Guckelberger, Olaf; Enjyoji, Keiichi
TI Thromboregulatory potential of endothelial **CD39**/nucleoside triphosphate diphosphohydrolase: modulation of purinergic signalling in platelets

SO Emerging Therapeutic Targets (2000), 4(2), 155-171
CODEN: ETTAF7; ISSN: 1460-0412

L8 ANSWER 23 OF 27 MEDLINE on STN DUPLICATE 7
AU Weksler B B
TI Antiplatelet agents in **stroke** prevention. combination therapy: present and future.
SO Cerebrovascular diseases (Basel, Switzerland), (2000) 10 Suppl 5 41-8.
Ref: 43
Journal code: 9100851. ISSN: 1015-9770.

L8 ANSWER 24 OF 27 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU McTaggart R A (Reprint); Broekman J; Peschon J; Stocking K; Choudhri T F; Kim L J; Connolly E S; Drosopoulos J H F; Maliszewski C R; Marcus A J; Pinsky D J
TI Cerebroprotective role of **CD39** (endothelial EctoADPase) in murine **stroke**
SO CIRCULATION, (2 NOV 1999) Vol. 100, No. 18, Supp. [S], pp. 1720-1720.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621.
ISSN: 0009-7322.

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AU McTaggart, Ryan A. [Reprint author]; Broekman, M. Johan; Peschon, Jacques; Stocking, Kim; Choudhri, Tanvir F.; Kim, Louis J.; Connolly, E. Sander, Jr.; Drosopoulos, Joan H. F.; Maliszewski, Charles R.; Marcus, Aaron J.; Pinsky, David J.
TI Cerebroprotective role of **CD39** (endothelial ectoADPase) in murine **stroke**.
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IN Maliszewski, Charles R.; Gayle, Richard B., III; Price, Virginia L.; Gimpel, Steven D.

TI **CD39** polypeptides as inhibitors of platelet activation and recruitment
SO PCT Int. Appl., 122 pp.
CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000023459	A1	20000427	WO 1999-US22955	19991013
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2345382	AA	20000427	CA 1999-2345382	19991013
AU 9964115	A1	20000508	AU 1999-64115	19991013
AU 772460	B2	20040429		
EP 1123306	A1	20010816	EP 1999-951733	19991013
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002527096	T2	20020827	JP 2000-577185	19991013
NZ 511488	A	20031031	NZ 1999-511488	19991013
US 2002002277	A1	20020103	US 2001-835147	20010413

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IN Maliszewski, Charles R.; Gayle, Richard B., III; Marcus, Aaron J.
TI Methods of inhibiting platelet activation and recruitment
SO PCT Int. Appl., 118 pp.
CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000023094	A2	20000427	WO 1999-US23641	19991013
WO 2000023094	A3	20000727		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9964256	A1	20000508	AU 1999-64256	19991013

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